Egyptian Journal of Anaesthesia (2014) **30**, 27–30

Egyptian Society of Anesthesiologists

Egyptian Journal of Anaesthesia

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Research Article

Use of lignocaine or nitroglycerine for blunting of hemodynamic stress response during electroconvulsive therapy

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Received 18 March 2013; revised 15 September 2013; accepted 26 September 2013
Available online 21 October 2013

KEYWORDS

Hemodynamic stress response;
Electroconvulsive therapy

Abstract *Background and aim of study:* Electroconvulsive therapy (ECT) is one of the safest methods used for the treatment of patients with mental illness today. It is associated with surge in heart rate and blood pressure for a brief period of time. However, as an extreme complication, the hemodynamic response to ECT can produce myocardial ischaemia and even infarction, as well as transient neurological ischaemic deficits, intracerebral haemorrhages, and cortical blindness. This study is aimed towards finding a reliable drug that can prevent this untoward hemodynamic response in immediate post-ECT period.

Place and duration of study: The study was conducted at Combined Military Hospital Skardu after permission from the hospital ethics committee from January 2011 to December 2011.

Study design: One thirty-four American society of Anaesthesiology (ASA) I & II patients of both genders were divided randomly in three groups named A, B and C. Patients were induced short general anaesthesia as per set protocol. Group A patients were given no additional drug, while group B patients had lignocaine 1 mg/kg and group C patients nitroglycerine (NTG) 3 μ/kg respectively just after induction. Heart rate (HR) and mean arterial pressure (MAP) were recorded 2 min after induction of anaesthesia just prior to ECT shock and then 1 min after ECT administration.

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Peer review under responsibility of Egyptian Society of Anesthesiologists.

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Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.egja.2013.09.004

Operation definitions: *Significant rise in heart rate* was defined as an increase in heart rate of 10 or more beats per minute after administration of ECT shock from baseline.
Significant rise in MAP was defined as the rise in MAP of 15 mm of Hg or more from the baseline after administration of ECT shock.
Results: Thirty-three (75%) of 44 patients in group A showed significant rise in HR as compared to group B where no patient showed a significant increase in HR ($p < .05$). In terms of MAP 29 (65%) out of 44 patients showed a significant rise in group A and 22 (52%) out of 42 patients in group B showed similar results showing statically insignificant difference between the groups. When comparing patients of groups A and C, only 11 (22%) out of 48 patients showed significant rise in HR and 13 (27%) patients showed significant rise in MAP. The difference was statistically significant in both variables ($p < .05$).
Conclusion: NTG provided more hemodynamic stability in post-ECT period as compared to lignocaine which only prevented a surge in HR without any effect on MAP. We conclude that NTG can safely be instituted for anaesthesia in ECT patients for prevention of hemodynamic stress response.

1. Introduction

Electroconvulsive Therapy (ECT) is one of the most effective therapy for the treatment of depressive episode severe, some cases of acute and chronic schizophrenia and bipolar affective disorder. Historically ECT was performed without anaesthesia, but currently it is performed under general anaesthesia with muscle relaxation. The aim of this brief general anaesthesia is to avoid the untoward effects of ECT without interfering with advantageous effects of ECT [1,2].

ECT is not free from its own hazards. Intense muscle contractions after ECT can lead to tongue bite, laryngospasm, bone fractures and joint dislocations. Autonomic nervous system stimulation causes bradycardia followed by tachycardia and hypertension. This can lead to cardiac arrest from bradyarrhythmias on the one hand and stroke and myocardial infarction in high risk patients due to tachycardia and hypertension. The hemodynamic response to ECT can produce myocardial ischaemia and even infarction, as well as transient neurological ischaemic deficits, intracerebral haemorrhages, and cortical blindness. In patients with severe cardiovascular disease, reduced intracranial compliance and vascular pathology, it may precipitate haemorrhage.

The aim of this study was to find an effective pharmacological agent to avoid acute tachycardia and hypertension in post-ECT period. Number of drugs can be used to prevent this untoward hemodynamic response, but we chose two conveniently available drugs in most operation theatre settings i.e. Nitroglycerin and Lignocaine.

Nitroglycerin (NTG) is a potent vasodilator traditionally used to treat angina attacks. It has a short half-life of 1–4 min. In the body, mitochondrial aldehyde dehydrogenase converts NTG into nitric oxide. NO increase the cGMP levels in the smooth muscles through which in turn reduces the free calcium concentration in the smooth muscle cell. The action is more pronounced on the veins causing blood pooling in this vessel compartment and thus prevents and treats hypertension [3]. The dose required is approximately 3 μg/kg.

Lignocaine is relatively short acting drug with elimination half-life of 90–120 min. It alters signal conduction in neurones by blocking voltage gated sodium channels in the neurone cell membrane that are responsible for signal propagation. With sufficient blockage the membrane of the postsynaptic neurone will not depolarise and will thus fail to transmit an action potential, thus blunting the sympathetic response [4]. The recommended dose for this purpose is 1–1.5 mg/kg.

2. Materials and methods

This study was conducted in the department of Anaesthesiology and department of Mental Health, Combined Military Hospital Skardu between January 2011 and December 2011. The study design was randomised controlled trial and was conducted after approval from hospital ethical committee. The hospital ethics committee stated that they have gone through the synopsis of the study and are permitting the department of anaesthesia and mental health to proceed with the study as it can benefit the patients undergoing ECT. The help of institution's biostatistician was acquired for the assessment of the appropriate sample size who used Altman's nomogram for the calculation of sample size. On this basis, 134 American Society of Anesthesiologist (ASA) physical status I & II patients were selected from those receiving general anaesthesia for ECT, and were divided into three groups. Groups were allocated using table randomisation. An independent observer was assigned to generate random allocation sequence. Patients with history of asthma, hypertension, heart blocks, recent MI/CVA (less than 6 weeks old), and severe osteoporosis were excluded from the study.

Patients allocated to groups A, B and C were 44, 42 and 48 respectively. The patients were randomly divided into three groups A, B and C. All were made NPO a per anaesthesia protocols. In operating room 18G cannula was passed and patients were given injection ondansetron 4 mg slow intravenous as an antiemetic. Induction was carried out with 2 mg/kg propofol and 1 mg/kg succinylcholine. Anaesthesia was maintained with 100% oxygen with facemask. In patients of group A no additional drug was given, in group B patients injection lignocaine 1 mg/kg was given and injection NTG 3 μg/kg was administered to patients of group C. HR and MAP were recorded 2 min after administration of anaesthesia just prior to ECT shock and 1 min after delivery of ECT shock.

An SR2 biphasic brief-pulse ECT machine was used to deliver the electrical stimulus via electrodes placed at patient forehead. Controlled or assisted ventilation was continued

with 100% oxygen until adequate spontaneous ventilation returned.

All statistical analysis was performed using SPSS version 20 and chi square test was used to determine the relation among interventions. A p value of < 0.05 was considered statistically significant.

2.1. Operation definitions

Significant rise in heart rate was defined as an increase in heart rate of 10 or more beats per minute after administration of ECT shock from baseline.

Significant rise in MAP was defined as the rise in MAP of 15 mm of Hg or more from the baseline after administration of ECT shock.

3. Results

The demographic data of patients in each group are shown in Table 1. Data showing pre- and post-ECT heart rate and mean arterial pressure in three groups is shown in the Tables 2 and 3 respectively.

Out of 44 patients in group A, 33 (75%) patients showed significant rise in heart rate, as compared to group B in which no significant rise in 1 min post-ECT heart rate was recorded. The p value was found to be less than 0.001 which was statistically significant. 29 (65%) patients of Group A showed significant rise in 1 min post-ECT MAP as compared to 22 (52%) patients of Group B ($p = 0.087$).

When comparing groups A and C, only 11 (22%) patients out of 48 in group C showed significant rise in 1 min post-ECT HR and 13 patients (27%) showed significant rise in 1 min post-ECT MAP. The difference was significant $p < 0.001$ in both cases showing better hemodynamic stability with NTG. The data are summarised in Tables 4 and 5.

Keeping aforesaid in view, it is inferred that NTG provided more hemodynamic stability in post-ECT period as compared to Lignocaine and no drug.

4. Discussion

Electroconvulsive Therapy (ECT) provokes a generalised seizure. This was first described in 1938 and was performed without anaesthesia for almost 30 years [5]. In recent years, ECT has presumed an increasingly significant role in the treatment for severe and medication resistant depression and mania, as well as in the treatment of schizophrenic patients with suicidal risk and catatonic symptoms [6]. Typically the acute phase of ECT is performed three times a week for 6–12 treatments. In successful cases, initial clinical improvement is usually evident after three to five treatments. Maintenance therapy can be performed at progressively increasing intervals from once a week to once a month to prevent relapses [7].

Understanding neurohormonal related physiological response of ECT is pivotal in providing anaesthesia to patients undergoing this procedure so that its beneficial effects can be spared while counteracting its adverse effects. Most modern ECT devices deliver a brief-pulse current, which is thought to cause fewer cognitive effects than the sine-wave currents which were originally used in ECT. Central nervous system seizure activity rather than electrical stimulus is responsible for the beneficial effect of ECT but the exact mechanism of the therapeutic effects is not yet understood. When an electric current is applied to brain via transcutaneous electrodes, the resultant electroencephalographic spike (EEG) and wave activity are accompanied by a generalised motor seizure and an acute cardiovascular response, which results in marked increase in cerebral blood flow and intracranial pressure [8]. However the magnitude of the acute hyperdynamic response to ECT appears to be independent of the duration of motor and EEG activity [9]. The hemodynamic response to ECT can produce myocardial ischaemia and even infarction [10], as well as transient neurological ischaemic deficits,

Table 1 Demographic data of patients in each group.

Group	Age (Years)	Sex	
		Male (n)	Female (n)
A ($n = 44$)	38 ± 10	19	25
B ($n = 42$)	34 ± 13	19	23
C ($n = 48$)	37 ± 9	23	25

Table 2 Comparison of Pre-ECT and 1 min post-ECT heart rates in different groups.

Group	Pre-ECT HR (beats/min)	Post-ECT HR (beats/min)
Group A ($n = 44$)	82.5 ± 14.4	91 ± 9.1
Group B ($n = 42$)	87.2 ± 11.8	83.5 ± 8.7
Group C ($n = 48$)	83.8 ± 10.6	88.6 ± 9.7

Table 3 Comparison of pre- and 1 min post-ECT MAP in different groups.

Group	Pre-ECT MAP (mmHg)	Post-ECT MAP (mmHg)
Group A ($n = 44$)	89.3 ± 16	112.8 ± 18.7
Group B ($n = 42$)	96.8 ± 16.6	113.9 ± 14.5
Group C ($n = 48$)	95.6 ± 16.3	106.5 ± 16.2

Table 4 Comparison of patients having significant rise in heart rate 1 min after ECT in three groups.

Group	Significant (Per cent)	Insignificant (Per cent)
A ($n = 44$)	33	11
B ($n = 42$)	0	42
C ($n = 48$)	11	37

Table 5 Comparison of patients having significant rise in MAP 1 min after ECT in three groups.

Group	Significant	Insignificant
A ($n = 44$)	29	15
B ($n = 42$)	22	20
C ($n = 48$)	13	35

intracerebral haemorrhages, and cortical blindness. Short term memory loss is common after ECT and more serious cognitive dysfunction have been described in the ECT literature, even though there is no scientific evidence of direct neurological evidence [11].

Hyperdynamic cardiovascular response that occurs after ECT is result of central activation of the autonomic nervous system. A brief parasympathetic discharge occurs immediately for the first 10–15 s after application of electrical current with a sympathetic discharge following within seconds. Within 10–12 s of the sympathetic surge caused by epinephrine and norepinephrine release, sinus tachycardia and arterial hypertension may develop. Plasma epinephrine increases to 15 times normal level, and plasma norepinephrine peaks can become three times higher than under normal resting conditions, with peak levels occurring within 60 s of electrical stimulation [12,13]. Systolic blood pressure is transiently increased by 30–40% and heart rate is increased by 20% or more, resulting in a two- to fourfold increase in the rate-pressure product, an index of myocardial consumption. Studies have shown that the concentration of epinephrine decreases towards normal values 5 min after ECT, and norepinephrine levels remain increased for twice as long. These hemodynamic changes produce an abrupt increase in myocardial oxygen consumption. It may be beneficial to administer drugs which blunt the hemodynamic stress response. There has always been a nagging thought in anaesthetist's mind to control this hemodynamic surge.

The purpose of our study was to blunt the hemodynamic insult associated with ECT to prevent the cardiovascular complications using a pharmacological agent. We used an adequate depth of general anaesthesia with brief muscle paralysis as our anaesthesia technique in the control group and then adding lignocaine and NTG in two different groups to find out whether these drugs provide an additional benefit on preventing hemodynamic surge associated with neurohormonal response of ECT. There are number of studies in which pharmacological management is instituted towards prevention of sympathetic surge in patients receiving ECT. However not much studies have specifically targeted the two drugs used in our research. In a study conducted at San Diego by Weigner MB et al. they concluded that lignocaine was not effective enough in attenuating the stress response associated with the ECT current [12]. Another study conducted by O Flaherty D et al. demonstrated that NTG produced better hemodynamic instability than esmolol [13].

Our study showed that lignocaine was effective enough to control the surge in HR but was unable to attenuate the rise in MAP in post-ECT period as compared to control group. On the other hand NTG was effective enough in controlling rise in both HR and MAP in immediate post-ECT period. We conclude that 3ug/kg NTG is superior to 1 mg/kg lignocaine in preventing hemodynamic surge in patients receiving ECT and practice can be instituted on regular basis in patients undergoing ECT procedure.

5. Limitations

This study is conducted at skardu which is a high altitude and can cause hemodynamic changes itself. The size of sample is small and results cannot be generalised on population.

Conflict of Interest

I declare that I have no significant competing financial, professional or personal interests that might have influenced the performance or presentation of this work described in this manuscript.

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